THE HEALTH EFFECTS OF ASBESTOS INHALATION

A Review of the Literature

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Introduction

The Massachusetts Commission on Asbestos was established in 1975 to investigate the public health hazards associated with the use of asbestos in schools and public buildings. Before embarking on its main investigation, the Commission finds it necessary to identify current research needs on the effects of low level asbestos inhalation over a period of several years. This report summarizes past studies relating to the occupational hazards, community effects, control and measurement of asbestos, so that informed recommendations can be made regarding the type of research still needed to support the Commission's activities.

Asbestos is used extensively by industry and individual consumers because of its unique combination of physical properties and ready availability at reasonable prices. Its fire and heat resistance makes it an excellent insulation material. It is also used in the manufacture of textiles, cement, and brake linings. There are, in the United States today, over one-hundred manufacturers of asbestos products.[1]

Several types of asbestos fibers are available commercially: chrysotile, crocidolite and amosite. In the United States, over 90% of the asbestos used by industry is chrysotile. The major producer is Quebec, Canada.[2]

Occupational Hazards

Four diseases have been linked to the inhalation of asbestos. They are pulmonary fibrosis, lung cancer, mesothelioma, and pleural calcification.

Asbestosis, or pulmonary fibrosis, was the first asbestos-related disease to be recognized by medical authorities. It is characterized by the formation of inactive, non-functioning fibrous scar tissue in the lung.[5] Extensive damage can eventually result in respiratory or cardiac failure. Symptoms include shortness of breath, finger clubbing and basal rales (abnormal breathing sounds).[7]

Although the existence of asbestosis as an occupational disease is now established, it was many years before the disease was widely recognized. In 1907, the first case of extensive lung scarring in a British textile worker was reported.[5] American recognition occurred twenty years later with the report of a miner who died of heart failure associated with fibrosis.[4]

These early communications and more recent epidemiologic studies highlight one important characteristic of asbestosis. It occurs mainly among those occupational groups experiencing heavy or moderate exposure. Extensive documentation in the United States has shown asbestosis to be common in the textile,[6] insulation,[8] and shipbuilding industries.[53] The exposure period is 20-40 years, with death following about two to 10 years later.[50]

The most common complication of asbestosis is cancer of the lung. The first case of lung cancer in a worker suffering from asbestosis was reported in 1935.[9] Later studies showed the incidence of cancer among asbestos occupations to be greater than selected control groups.[10,11,12] A British researcher, in fact, reported that textile workers incurred a 10-times higher risk of having lung cancer than the general population.[13]

Cancer has been specifically linked to chrysotile fibers, the most common type used in the United States. Chrysotile miners[19] and building product workers[14,16] died of respiratory cancers at a greater rate than would have been anticipated based on general mortality rates.

Recent evidence indicates that the dangers of asbestos-related cancers will increase dramatically in the future. An epidemiologic study of Dresden, Germany, asbestos workers indicated that lung cancers became more common as observation continued. This was due, in part, to the longer latency period required for development of cancerous tumors compared to pulmonary asbestosis. Researchers found that death from pulmonary asbestosis occurred at an average of 25.7 years from onset of exposure, whereas 30.7 years was required for the development of cancer.[15]

An additional complication is provided by the recent revelations indicating a co-carcinogenic or synergistic effect with cigarette smoking. Calculations by New York City scientists indicate that asbestos workers who smoke have about ninety-two times the risk of dying from lung cancer as individuals who neither

work with asbestos nor smoke.[17] A disturbing sidelight is that other synergistic effects may come to light in the future.

Malignant tumors of the lung or chest cavity, known as mesothelioma, were recently regarded as very rare. In 1960, 33 cases of pleural mesothelioma were described in a South African mining town. All but one of the exposures was linked to the inhalation of crocidolite asbestos.[21] Five years later, mesothelioma was linked to chrysotile asbestos in the United States[24] and the Netherlands.[30]

It soon became apparent that mesothelioma also posed a substantial threat to individuals experiencing low exposure levels. In Canada, mesothelioma was found more common in insulation workers than miners.[31] In London and Cape Town, a higher-than-expected incidence of mesotheliomas was found in people living near asbestos factories and fields.[26,28] This was verified in New Jersey, where researchers found mesothelioma cases near an asbestos mill. A comparison between cancer and mesothelioma deaths showed people with mesothelioma dying at a younger age, thereby lending support to the low-level exposure theory.[32] Initial exposure to asbestos occurred 25 to 38 years before death from mesothelioma.[24,25,33] Thus far, there have been few cases reported among the general population. A review of several thousand deaths in the United States revealed only three mesothelioma cases.[47] To complicate matters, asbestos is not the only cause of mesothelioma; it has also been produced by silica[22] and polyurethane.[23]

Another disease often associated with low exposure to asbestos is pleural calcification, the depositing of insoluable calcium salts in the lining of the lung. Epidemiologists found pleural calcification in Finnish farmers living near an asbestos mine[34] and Bulgarian agricultural workers in an asbestos-rich soil region.[35] New York insulation workers also showed signs of calcification 20 years after their initial exposure.[36]

A potential hazard to children has been demonstrated. Aggravation of pulmonary disease has been the major finding among children living near an asbestos plant.[37,39]

Effects on the General Community

Research concerning the health effects of asbestos on the general public has been focused in two areas. The first has been a determination of the prevalence of "asbestos bodies" in the lungs of urban dwellers. The second has been a measurement of asbestos contamination during and after building construction.

The presence of "asbestos bodies" in the lungs of occupationally exposed groups has been acknowledged for some time.[6,40] It was not until 1960, however, that it was realized how widespread the existence was in the general population. Pathologists found 26.4% of lung smears from 500 consecutive autopsies in Cape Town, South Africa, contained these "bodies."[41] A more recent study in Pittsburgh found the percentage of "ferruginous bodies" to be as high as 98.[48] These researchers preferred to use the term "ferruginous bodies" because optical microscopy techniques do

not permit positive identification of asbestos fibers in these bodies. They demonstrated that other materials, such as cosmetic talc and glass fibers, can produce ferruginous bodies indistinguishable from those produced by asbestos.[46] Positive identification of an asbestos fiber core can only occur with high magnification electron microscopy. Using this technique, chrysotile asbestos was identified in 28 consecutive samples of lung tissue in New York City. Most of the asbestos was present as fibrils (thin fibers 200-400 angstroms in diameter). Due to its brittleness, chrysotile tends to split into these fibrils when inhaled.[54] It is estimated that 200 to 1,000 fibers and fibrils can be seen by electron microscopy for every fiber identified by optical microscopy.[64]

The deleterious effect of these short and thin fibers has not yet been determined. Evidence exists to suggest both a harmful and non-harmful consequence.[43,62] J.G. Thomson, in explaining his views on the harmful aspects of short-fibered asbestos, concluded

> "asbestos fibers of this diameter as an air contaminant in cities of significant degree now or in the future would constitute a menace against which man would be relatively powerless."[45]

The source of these fibers are the many asbestos-containing products being used in our society. One of these, asbestos fireproofing, was used extensively in high-rise office buildings from 1958 until its ban in 1973. In 1970, alone, more than 40,000 tons of asbestos material were used for this purpose.[57] Measurements of asbestos emissions during and after the application of fireproofing material have been conducted.

It was estimated that 15 tons of asbestos were emitted from these materials during building construction in 1968.[52] Asbestos fiber concentrations at the construction sites ranged from 30 to 100 fibers per cubic centimeter. (The current government standard is two fibers per cubic centimeter.) Concentrations were still high 75 feet away from the operation.[57]

After construction ends, asbestos levels can still be detected. Mass analysis techniques showed the average asbestos levels in 19 United States buildings to be between 2.5 and 200 nanograms per cubic meter. (A nanogram is equivalent to 10^{-9} grams.) Concentrations above 100 nanograms/cubic meter were considered indicative of a potential harm, because this was the level found in the homes of asbestos-insulation workers (the source is presumed to be the dust brought home in the workers overalls). The fibrous spray-on fireproofing was found to contribute more towards pollution than cementitious fireproofing.[65]

In contrast, fiber counting procedures were employed during a British survey of 60 buildings, including schools, offices, and residences, in which asbestos was used for insulation, fire-proofing, and/or other application. In over 90% of the locations sampled, the asbestos dust concentrations did not exceed one-tenth of the level regarded as acceptable for occupational exposure. More than 40% of the concentrations were of the same order as the level in buildings where no asbestos had been used.[51]

It is difficult to compare these two surveys. The methods used are different. The British survey relied on fiber

counts; the American study did not. The American scientists considered small particles and fibers in their mass determinations; the British excluded fibers less than five microns in length. Neither group of researchers provided information on when their asbestos samples were taken. Samples taken during business hours will show more fibers than those taken at night as a result of the movement of people causing air currents that circulate the fibers.

Ambient concentrations have also been measured. Researchers in New York City found chrysotile concentrations ranging from 10 to 50 nanograms per cubic meter,[58] and European investigators found asbestos fibrils present in all the towns surveyed.[59]

Measurement and Control of Asbestos Emissions

Measures to control asbestos contamination of the environment have been pursued by both the government and private individuals. Federal oversight of the problem is centralized in the Occupational Safety and Health Administration (OSHA) and the Environmental Protection Agency (EPA).

OSHA regulates airborne concentrations in the workplace according to the threshold concept. Measurements above the threshold level are considered harmful; measurements below are judged safe. The current regulation designates two fibers per cubic centimeter (2f/cc) as the threshold.[67] Consideration is currently being given to a more stringent level of 0.5 f/cc.[73] Optical microscope analyses used permit identification of only those fibers longer than five microns.

Alternative measurement procedures have been proposed. A threshold level of 30 nanograms/cubic meter has been proposed by Connecticut environmental protection authorities.[74] This mass analysis technique, however, is biased in favor of small fibers[66] and agreement has not yet been reached on the risks associated with small fibers (see previous section). Other methods include measurement of asbestos particles (impinger analysis), and small fibers by electron microscope. Each method measures something different, and therefore, they do not correlate well.[66]

Ultimate revision of the current standard may result in the establishment of a zero-base level. The National Institute for Occupational Safety and Health has stated that present data is insufficient to set a standard for long-term asbestos exposure other than zero which could preclude development of malagnancies.[67] In fact, recent disclosures indicate that the proposed level of 0.5 f/cc may still be too high. Scientists have found the risk of respiratory disease to be three times the expected rate for miners exposed to fiber concentration levels of 0.24 f/cc.[72]

The EPA's control of ambient asbestos levels is aimed at reducing the danger to the public from the use of asbestos products. No numerical standards have been promulgated because of the difficulty encountered in prescribing a standard with an ample margin of safety.[68] The current regulations, as applied to asbestos spraying, require that "materials used to insulate or fireproof buildings, structures, pipes and conduits shall contain less than 1% asbestos.[68] As a result, asbestos insulation is being replaced by fiber glass.[70]

Private individuals have also taken steps to control asbestos where a problem had been identified. Yale,[75] UCLA,[75] and a Wyoming elementary school[65] have all taken measures to remove asbestos from their buildings.

Yale found asbestos emissions in their Art and Architecture Building exceeded the 2.0 f/cc limit in some instances. All building users were exposed to a level over that found in the ambient air.[75]

Conclusion

The preceding review of the asbestos literature has isolated several avenues of research that could be pursued by the Massachusetts Asbestos Commission to obtain basic information desirable in executing its mandate. The suggested areas of research are:

(1) The merits of a zero base level of asbestos emissions

(2) The health hazards of low-level asbestos pollution. Studies on ambient concentrations have only measured levels without searching for epidemiologic evidence of mortality associated with such levels.

(3) Establishment of a uniform measurement technique.

(4) The dangers of inhaling short-fibered asbestos.

(5) Studies of children to determine any immediate or long-term hazards.

(6) Technical procedures to minimize emissions (a filtering device is one possibility).

Most scientists agree to the danger of asbestos in the occupational environment. They are, however, divided on a safe exposure limit. Each year, reports suggest lower threshold levels as new evidence becomes available. The merits of a zero-base standard are currently under consideration.

The determination of potential harm to the general public has not been an easy task. Epidemiologic studies have noted an increased health risk of mesothelioma and pleural calcification to those people living near asbestos mines and factories, but no clear-cut evidence has arisen to implicate a danger situation in our cities and towns.

Previous research has been limited to the measurement of "asbestos bodies" and fibers present in the lungs of urban dwellers, and emissions from buildings. Some of these studies have used the occupational standards as guidelines. Such procedures have tended to underestimate the asbestos contamination of our ambient air, since occupational levels will always be higher. Whether the lower urban concentrations are harmful remains open to debate. No researcher has found a definitive link between asbestos and increased mortality. This does not mean there is none. One can easily imagine the difficulties encountered in a thorough survey of the general population. There are so many factors to consider that meaningful conclusions are difficult to reach. Research is complicated by the co-carcinogenic effect of asbestos inhalation. It has been demonstrated smoking and asbestos result in increased risks. Can we carry the argument one step further and say Americans suffering from any kind of respiratory ailment

(asthma or bronchitis, for example), incur an increased risk? Such a statement is not possible at the present time, but is indeed within the realm of possibilities.

A new measurement technique would be helpful in reaching a consensus on the harm to the general public. Some researchers have been measuring urban asbestos concentrations by mass analyses instead of fiber counts (used by OSHA). Circumstantial evidence indicates that a level exceeding 30 nanograms per cubic meter is harmful. Efforts to establish one measurement procedure would greatly enhance medical research. The acceptance and use of one procedure would permit easier comparison of medical data and expedient development of standards. Mass analysis, using electron microscopes, has its disadvantages. Because it is costly and time consuming, extensive use is precluded. Development of a new method may be needed.

An additional question that has arisen, in the context of ambient asbestos levels, is the harm of short fibers. Most of the fibers found in the air we breathe are invisible under the optical microscope. Electron microscopes are needed to detect the short fibrils. The harm of these fibrils is currently being debated.

Studies of children have been scarce. We do not know if high asbestos concentrations pose a danger to these young members of our community. The only in-depth examination was conducted in 1958. Soviet investigators found some reason for alarm when they noted increased respiratory ailments (pneumonia) among children living near an asbestos factory.

While medical questions remain unsettled, precautions can still be taken. As reported, some private individuals have taken measures to remove asbestos insulation at great financial expense. A less costly method of asbestos containment may be feasible and research should be undertaken to explore various possibilities. One alternative, a filtering device, has not been extensively tested.

Asbestos is an important material in our society, yet it has the potential for great harm. A balance must, therefore, be reached between its beneficial and deleterious aspects. With additional research in the areas outlined above, we can establish workable and lasting regulations.

Although this report was prepared for the Massachusetts Commission on Asbestos, the hazards of asbestos are not limited to the Commonwealth. Instances of asbestos contamination have been documented in many sections of the United States, and the widespread use of asbestos-containing products makes the potential for increased contamination an issue that should be one of national concern. Thus, the implications of the Commission's research and deliberations should not stop at the borders of Massachusetts.

ANNOTATED BIBLIOGRAPHY

I. Background information

1. O. Bowles, Asbestos-A Materials Survey, Bureau of Mines Information, Circ. 7880, 1959.

   This report contains information on the production and trade aspects of asbestos. A discussion of the exploration, mining, milling, and processing methods are included with an extended presentation on the structure of the major industries.

2. N.W. Hendry, "The Geology, Occurrences and Major Uses of Asbestos," Ann. N.Y. Acad. Sci., 132:12 (1965).

   A discussion of the major types and uses of asbestos fibers is presented.

II. Asbestosis

3. W.E. Cooke, "Pulmonary Asbestosis," Brit. Med. J. 2:1024(1927).

   A woman, aged 33, started work at the age of 13 in an asbestos factory in which no provision was made for the extraction of dust. X-rays after death showed fibrosis in her right lung.

4. R.G. Mills, "Pulmonary Asbestosis, Report of a Case," Minn. Medicine, 13:495(1930).

   This represents the first recorded case of asbestosis in the United States. The disease lasted for 17 years and the patient's first contact with asbestos was 32 years before his death when he worked at a South American asbestos mine.

5. E.R.A. Merewether, "A Memorandum on Asbestosis," Tubercle, 15:69 (November 1933).

   A summary of the medical literature and British regulations on asbestosis is presented. A very good description of the disease's symptoms is included.

6. W.C. Dreesen, J.M. Dallavale, T.I. Edwards, J.W. Miller, and R.R. Sayers, A Study of Asbestos in the Asbestos Textile Industry, Public Health Bulletin 241, Washington D.C.: U.S. Government Printing Office, 1938.

   Four textile plants were surveyed. The incidence of asbestosis increased with increasing dust exposure. Clear cut cases were found in dust concentrations above five million particles per cubic foot (5 mppcf).

7. H.B. Eisenstadt, "Benign Asbestos Pleurisy," Journal of the AMA. 192:419 (May 3, 1965).

   The stages in the development of asbestosis are described. Following a latency period of 10 or more years, respiratory symptoms appear. As the disease advances, systemic complaints are added, such as fatigue and weight loss. Finally extensive destruction of lung tissue leads to respiratory and cardiac failure.

8. I.J. Selikoff, J. Churg, and E.C. Hammond, "The Occurrence of Asbestosis among Insulation Workers in the United States," Ann. N.Y. Acad. Sci., 132:139(1965).

   An investigation involving 1522 asbestos insulation workers in the N.Y.-N.J. metropolitan area was conducted. Among 392 individuals examined more than 20 years from onset of exposure, radiological evidence of asbestosis was found in 339. In individuals with less than 20 years of exposure, radiological evidence of asbestosis was less frequent. It is concluded that asbestosis and its complications are significant hazards among U.S. insulation workers.

III. Lung Cancer

9. K. Lynch and W.A. Smith, "Pulmonary Asbestosis: Carcinoma of Lung in Asbesto-Silicosis," Amer. J. Cancer, 24:56 (1935).

   The case history of a 57-year old man who died of lung cancer is discussed. This man had suffered long occupational exposure to dust. He worked in a cotton mill for 22 years and an asbestos factory for 21 years. He suffered from extensive fibrosis before contracting carcinoma.

10. E.R.A. Merewether, Annual Report of the Chief Inspector of Factories for the Year 1947, London: H.M. Stationery Office, 1949.

    Lung cancer was reported at necropsy in 13.2% of cases with asbestosis (31 out of 235) but in only 1.3% of cases with silicosis.

11. F. Homburger, "The co-incidence of primary carcinoma of the Lungs and Pulmonary Asbestosis," Amer. J. Path 19:797(1943).

    Over a 20-year period, eight cases of asbestosis were found in 4,137 autopsies at the Yale Medical School. Of these eight cases, four were associated with lung cancer (50%). In contrast, lung cancer was found in only two (12%) of 17 cases of silicosis.

12. S.R. Gloyne, "Pneumoconiosis: A histologic survey of necropsy material in 1205 cases," Lancet, 1:810 (1951).

    During the period 1929-49, the London Chest Hospital conducted microscopical examinations to determine the presence of pulmonary disease attributable to the inhalation of dust. Among the sample examined, 132 cases were asbestos workers. 121 suffered from asbestosis and 17 of these asbestosis cases died of lung cancer (14.1%). Only 6.9% (55/796) of silicosis patients died of lung cancer.

13. R.O.Doll, "Mortality from lung cancer in asbestos workers," Brit.J.Ind.Med., 12:81(1955).

    The medical history of 113 men who worked for at least 20 years in places where they were liable to be exposed to asbestos dust was compared with the expected number based on the mortality experience of the whole male population. 39 deaths occurred in the group. 15.4 were expected. The excess was due to lung cancer associated with asbestosis. Duration of exposure ranged from 13 to 32 years.

14. I.J. Selikoff, J. Churg, E.C. Hammond, "Asbestos Exposure and Neoplasia," Journal of the AMA, 188:22(1964).

    A study of 632 insulation workers from the New York - New Jersey area revealed that of 255 deaths, 45 were due to lung cancer. Based on lung cancer rates of the United States white male population, 6.6 were expected.

15. G. Jacob and M. Anspach, "Dresden Asbestos Workers," Annals N.Y. Acad. Sci., 132:536(1965)

Initial studies in the Dresden area showed no increased incidence of lung cancer above that experienced by the general population. However, in a follow-up conducted from 1958-64, a sharp increase was noted. Twice the expected number was encountered among men and 10 times the expected number among women. The long latency period for cancer was given as one reason.

16. P.Enterline and M.A. Kendrick, "Asbestos-Dust Exposures at Various Levels and Mortality," Archives Env. Health, 15:181(1967).

A study was conducted on the mortality experience of 21,755 white males who worked in three asbestos product industries. Comparisons were made with the mortality experience of the entire United States white male population. Only in the textile industry was an elevated death rate for all causes found. Asbestos building product and friction materials workers had increased lung cancer and asbestosis rates.

17. I.J. Selikoff, E.C. Hammond and J.Churg, "Asbestos Exposure, Smoking and Neoplasm, Journal of the AMA, 204:104(1968).

Observation of 370 insulation workers from 1963 to 1967 revealed an increased risk of lung cancer among smokers. Their calculations suggested that asbestos workers who smoke have about 92 times the risk of dying from lung cancer as men who neither work with asbestos nor smoke. It was also determined that an asbestos worker who smoked had eight times the risk of dying compared with cigarette smokers having no asbestos exposure.

18. G. Berry, M.L. Newhouse, M. Turok, "Combined Effect of Asbestos Exposure and Smoking on Mortality from Lung Cancer in Factory Workers," Lancet, 2:476-8 (September 2, 1972).

The smoking habits of over 1,300 male and 480 female asbestos factory workers were studies and their mortality from lung cancer over a 10 year period examined. A significant excess of deaths was found among workers who were severely exposed and who smoked. No excess was found with low or moderate exposure, whether smokers or non-smokers.

19. J. Corbett McDonald, M.R. Becklake, G. Gibbs, A.D. McDonald, C.E. Rossiter, "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," Arch.Env.Health, 28:61 (Feb. 1974).

A follow-up study on Quebec miners suggested that mortality from respiratory cancer was 50% above expectation.

IV. Mesothelioma

20. G. Bonser, J.S. Faulds, M.J. Stewart, "Occupational Cancer of the Urinary Bladder in Dyestuffs Operatives and of the Lung in Asbestos Textile Workers and Iron Ore Miners," Amer. J. Clinical Path, 25:126 (1955).

    A survey of 72 asbestos textile workers who died from asbestosis was analyzed to determine further complications. 40 had tuberculosis, 14 had lung cancer and four had mesothelial tumors.

21. J.C. Wagner, C.A. Sleggs and P. Marchand, "Diffuse Pleural Mesotheliona and Asbestos Exposure in the Northwestern Cape Province," Brit.J. of Ind. Med., 17:260(1960).

    Thirty-three cases of mesothelioma are described, all but one having a probable exposure to crocidolite asbestos. This exposure occurred in the Asbestos Hills in northwest Cape Town, South Africa.

22. J.C. Wagner, "Experimental Production of Mesothelioma Tumors of the Pleura by Implementation of Dusts in Laboratory Animals," Nature, 196(4850):180(Oct.13, 1962).

    Mesothelial tumors were produced by silica as well as chrysotile and crocidolite asbestos in rats.

23. W.C. Hueper, "Cancer Induction by Polyurethane and Polysilicone Plastics," J. Natl. Cancer Institute, 33:1005(1964).

    Polyurethane can cause mesothelioma in rats.

24. I.J. Selikoff, J. Churg, E.C. Hammond, "Relation between Exposure to Asbestos and Mesothelioma," New England Journal of Medicone, 272:560(1965).

    Three studies were conducted to determine the harm of chrysotile asbestos. A review of 2500 consecutive autopsies in the United States from 1953-64 revealed 26 asbestosis cases with seven mesotheliomas. An examination of Armed Forces files found 12 of 45 mesotheliomas contained asbestos bodies. A survey of insulation workers found 17% lung cancers and 3.2% mesotheliomas among 307 consecutive deaths.

25. M.L. Newhouse and H. Thompson, "Epidemiology of Mesothelial Tumors in the London area," Annals N.Y. Acad. Sci. 132:579(1965).

Two groups of patients who attended a large hospital in London were examined to determine their exposure to asbestos. One group consisted of patients who died from mesothelioma. The other group served as a control. Of the patients suffering from mesothelioma, 56.2% had been exposed to asbestos, as compared to 11.8% of the control series.

26. J.C. Wagner, "Diffuse Mesothelial Tumors," Ann. N.Y. Acad.Sci., 132:575(1965).

A brief historical sketch is presented citing efforts of the British Medical Research Council to uncover a link between asbestos contact and the development of mesothelioma. By the end of 1961, the Council had diagnosed a total of 87 mesotheliomas in Cape Town, South Africa and its vicinity. 12 of the 87 cases had definite industrial exposure. The remainder lived in the regions surrounding the asbestos fields.

27. D.O'B. Hourihane, "A Biopsy Series of Mesotheliomata," Ann. N.Y. Acad. Sci., 132:647(1965).

A survey of histological material at a single hospital revealed 84 acceptable mesotheliomata cases which had been diagnosed over a period of 10 years. Asbestos bodies were found in the lungs or sputum of the majority of these patients.

28. M.L. Newhouse and H.Thompson, "Mesothelioma of Pleura and Peritoneum following Exposure to Asbestos in the London Area," Brit. J. Ind. Med., 22:261(1965).

The study outlined in reference 25 is explained in terms of neighborhood exposures. Among those with no evidence of occupational or domestic exposure, 30.6% of the mesothelioma patients lived within one-half mile of an asbestos factory. The interval between first exposure and development of mesothelial tumors ranged from 16 to 55 years.

29. M. Borow, A. Conston, L. Livernese and N. Schalet, "Mesothelioma and its Association with Asbestosis," Journal of the AMH, 201:587 (Aug. 21, 1967).

A review of the autopsy statistics in a local New Jersey hospital over a 10-year period (1955-65) found 22 cases of lung cancer associated with asbestos as well as 17

cases of mesothelioma. Two of the mesothelioma cases contracted their illness from only environmental exposure. The hospital is near an asbestos mill.

30. H.T. Plandeydt, "Asbestos and Mesothelioma in the Netherlands," TNO-Nieuws, 27(11):667(Nov.1972).

In a statistical study of mesothelioma in the Netherlands, all histologically verified cases of mesothelioma were registered starting from January 1969. Chrysotile was present in all (28) of the cases. This finding has significance concerning low level exposure because the Netherlands does not mine its own asbestos. All the cases, therefore, resulted from limited occupational exposure (shipyard work) or environmental contamination. A subsequent study of harbors and shipyards in the Rotterdam area demonstrated an occupational exposure to asbestos in nearly all mesothelioma cases.

31. A.D. McDonald, and J. Corbett McDonald, "Epidemiologic Surveillance of Mesothelioma in Canada," Can.Med.Assoc.J., 109(5):359-362 (September 1, 1973).

The number of fatal malignant mesotheliomas occurring in Canada during the period 1960-1970 was ascertained by contacting all Canadian pathologists. A history of definite or probable occupational exposure was found in 30% of male cases and 11% of female cases. The manufacture of asbestos products or insulation materials was associated with more mesotheliomas than mining and milling. No case, other than those occupationally or domestically exposed had lived within 20 miles of an asbestos mill or mine. (An early report of this study is found in Cancer, 26:914 (1970).)

32. M.Borow, A. Conston, L.Livernese and N.Schalet, "Mesothelioma following Exposure to Asbestos: A Review of 72 Cases," Chest, 64:641 (Nov. 1973).

A review of a previous study (see reference 29) has uncovered 72 mesothelioma cases near a New Jersey asbestos mill. The incidence was found to be increasing each year. A discussion of low exposures and casualty with asbestos inhalation is included.

33. M. Greenberg and T.A. Lloyd Davies, "Mesothelioma Register: 1967-1968," Brit. J. Ind. Med., 31:91-104(1974).

246 definite cases of mesothelioma were identified in 1967-68 by British authorities. Occupational exposure to asbestos was found in 68% of the definite cases. The interval between first exposure and death was 25 years. The duration of exposure varied. In one-eighth of the cases, it was under five years.

V. Pleural Calcification

34. R. Kiviluoto, "Pleural Calcification as a Roentgenologic Sign of Non-occupational Endemic Anthophyllite-Asbestosis" Acta Radiol. Suppl., 194:1 (1960).

Four hundred ninety-nine instances of calcification were uncovered during a community x-ray survey among 6,312 adults in a Finnish commune. A nearby commune had no cases among 7,101 adults. The main difference was the presence of a nearby asbestos mine in the first case.

35. C. Zolov, T. Bourilkov, and L. Babadjov, "Pleural asbestosis in agricultural workers," Env. Research, 1:287 (1967).

The same rate of pleural calcification was found in Bulgarian farmers and miners. The farmer's increased rate of illness was linked to asbestos-rich soil.

36. I.J. Selikoff, "The Occurrence of Pleural Calcification among Asbestos Insulation Workers," Ann. N.Y. Acad. Sci., 132:351(1965).

An x-ray study of 1,117 asbestos insulation workers showed pleural calcification to be a common finding. Altogether, 150 instances of such calcification were present. Calcification generally takes place 20 years after initial exposure to asbestos.

VI. Asbestos and Children

37. A.T. Bobyleva, R.M. Bukhantseva, S.E. Lovtsova and M.S. Sadilova, "Dust Concentration in Residential Districts of Asbest City and Its Effect on the Children's Health," Gigiena i Sanit., 23(11):9(1958).

Data collected in Asbest City (in Sverdlovsk, USSR) showed that factory-emitted asbestos dust was the primary cause of a sharply defined air pollution area extending three kilometers out from the plant. An increased incidence of pneumonia was demonstrated among children.

38. G.W. Grundy, R.W. Miller, "Malignant Mesothelioma in Childhood," Cancer, 30:1216(1970).

A review of 13 cases of childhood mesothelioma between 1960 and 1968 did not allow determination of possible asbestos exposure because case histories had no information on environmental exposures. It is recommended that practitioners obtain better environmental histories in future cases.

39. P. Wehrle, D. Hammer, Summary Report: Illnesses of Children, Air Pollution Medical Research Conference, AMA, Chicago, 1974 (?).

Adverse effects to children from proximity to an asbestos plant have been documented. Aggravation of pulmonary disease and lower respiratory diseases were the major clinical findings.

VII. Effects on the General Community

40. K.Lynch and W.A. Smith, "Asbestos Bodies in Sputum and Lung," Journal of the AMA, 95:659(1930)

Examination of the sputum of four asbestos workers revealed the presence of asbestos bodies.

41. J.G. Thomson, "Asbestos and the Urban Dweller," Ann. N.Y. Acad. Sci., 132:196(1965).

The extent of asbestos fiber inhalation by urban dwellers was investigated by counting asbestos bodies in lung smears from 500 consecutive autopsies in subjects over the age of 15 in Cape Town, South Africa, and 500 in Miami, Florida. The results were similar in the two cities. No less than 30% of the males and 20% of the females showed asbestos bodies. In 85% of the positive cases, the bodies were scanty, were not associated with pulmonary changes, and were regarded as a result of urban air contamination.

42. D. Cauna, R.S. Totten, and P. Gross, "Asbestos Bodies in Human Lungs at Autopsy," Journal of the AMA, 192:371 (May 3, 1965).

The incidence of asbestos bodies in the lungs was investigated in 100 autopsies of Pittsburgh adults. Asbestos bodies were found in 41% of the cases. They were not encountered in persons below 24 years old. Pulmonary fibrosis was found in two cases; mesothelioma was not encountered. Primary lung carcinoma occurred in one patient with asbestos bodies and one without.

43. J.M.G. Davies, "Electron Microscope Studies of Asbestosis in Man and Animals," Ann. N.Y. Acad. Sci., 132:98(1965).

Electron microscope studies show that the bulk of the asbestos dust that gets into human and guinea pig lungs is less than one micron in length. Preliminary results of animal studies suggest that these small fibers may be just as harmful as large fibers.

44. L. Anjilvel, W.B. Thurlbeck, "The Incidence of Asbestos Bodies in the Lungs at Random Necropsies in Montreal," Can. Med. Assoc. J., 95:1179 (1966).

The incidence of asbestos bodies in the lungs of adult patients selected at random, who died in four Montreal hospitals, was studied by examining fresh, unstained smears of lungs obtained at necropsy. Asbestos bodies were present in 48% (57% of the men and 34% of the women). No link between the bodies and those patients with malignant diseases was established.

45. J.G. Thomson, W.M. Graves, "Asbestos as an Urban Air Contaminant," Arch. Path, 81:458 (May 1966).

A review of the Miami data on asbestos bodies (see reference 41) is presented with an assessment of the study's impact. The extensive use of asbestos products capable of emissions and the danger of small fibers is mentioned.

46. P. Gross, "Pulmonary Ferruginous Bodies," Arch. Path. 85:539(1968).

All fibrous dusts do not share the ability of asbestos to produce lung damage. Such materials as fibrous aluminum silicate, silicon carbide, whiskers, cosmetic talc and glass fibers produce ferruginous bodies experimentally which are indistinguishable from those produced by asbestos fibers.

47. I.J. Silikoff and E.C. Hammond, "Community Effects of Non-occupational Environmental Asbestos Exposure," Amer. J. of Public Health, 58(9):1658 (Sept. 1968)

A review of 31,652 consecutive deaths in the general population (1959-62) revealed only three cases of mesothelioma. Preliminary results of an autopsy study of New York City residents showed 50% containing asbestos bodies.

48. M. Utidjiam, P. Gross, R. deTreville, "Ferruginous Bodies in Human Lungs," Arch Env. Health, 17:327 (Sept. 1968).

Ferruginous bodies with a transparent central core or no visible central filament (indicative of asbestos) were present in 97 of 100 autopsies; they were found in 98% of the men and 95.5% of the women. Thirty-two of the patients in the series had some form of malignant disease but there was no association between the malagnancies and the abundance of asbestos bodies.

49. P. Gross, "Pulmonary Ferruginous Bodies in City Dwellers" Arch. Env. Health, 19(2):186 (Aug. 1969).

On the basis of its unique electron diffraction pattern, chrysotile was decisively excluded as a constituent in the core of all (28) ferruginous bodies isolated from lungs of urban dwellers in Pittsburgh.

50. R.J. Sullivan and Y.C. Athanassidas, Air Pollution Aspects of Asbestos, U.S. Department of Commerce, National Technical Information Service NTIS #PB 188 080, 1969.

This report provides an excellent technical summary of the asbestos literature. A discussion of animal studies reveals that asbestosis and cancer have been experimentally produced after exposure to asbestos.

51. J.C. Byron, A.A. Hodgson and S. Holmes, "Dust Survey Carried Out in Buildings Incorporating Asbestos - based Materials in their Construction," Ann. Occ. Hyg. 12:141-5, 1969.

An assessment has been made of the amount of asbestos dust in the respirable size range in typical situations where the principal type of asbestos-based building materials have been used. In over 90% of the sixty buildings surveyed, the asbestos dust concentrations were less than one-tenth of the level regarded as acceptable for occupational exposure.

52. W.E. Davis, National Inventory of Source & Emmissions: Asbestos - 1968, APTD-70, EPA Research Triangle Park, N.C., Feb. 1970.

Estimates of emissions are made for various asbestos products in 1968. Emissions from the use of spray-on fireproofing are estimated at 15 tons, based on an emissions factor of 10 pounds per ton of asbestos applied.

53. R.L. Murphy, G.C. Ferris, W.A. Burgess, J. Worcester and E.A. Gaenster, "Effects of Low Concentrations of Asbestos," N. Eng. J. Med., 285:1271-8(1971)

A survey of 101 pipe coverers involved in new ship construction revealed an increased risk of asbestosis. The disease was first found after 13 years of exposure or 60 million particles per cubic foot-years. The prevalence was 38% after 20 years.

54. A.M. Langer, I.J. Selikoff, A. Sastre, "Chrysotile Asbestos in the Lungs of Persons in New York City," Arch. Env. Health, 22:348(1971).

When inhaled, chrysotile tends to split into unit fibrils, 200-400 Angstroms in diameter, invisible with

55. P. Gross and R. de Treville, "Inorganic Fibers in the Lungs of Urbanites: Are They a Health Hazard," paper presented to the Amer. Ind. Hygiene Assoc. Annual Conf., May 24-8, 1971 (Toronto, Canada).

A good summary of the debate surrounding asbestos in the lungs of urban dwellers is presented. It is estimated there are 8 to 80 million inorganic optically visible fibers in the lungs of adult city dwellers.

56. A.M. Langer, V. Baden, E.C. Hammond and I.J. Selikoff, "Inorganic Fibers Including Chrysotile, in Lungs at Autopsy," Inhaled Particles III, ed. by W.H. Walton, British Occ. Hygiene Society, vol. 2:683-94, 1971.

Microscopic evaluation of lung sections in 3000 consecutive autopsies of New York City residents showed asbestos bodies present in 48.3%. Electron microscope analysis can only provide positive identification of chrysotile in these bodies. Twenty-eight consecutive cases showed chrysotile fibers or fibrils present. In 1038 cases, thin inorganic fibers were found.

57. W.B. Reitze, W.J. Nicholson, D.A. Holiday, I.J. Selikoff, "Application of Sprayed Inorganic Fiber-Containing Asbestos: Occupational Health Hazards," Amer. Ind. Hyg. Assoc. J., 33:178 (March 1972).

Contains an extensive overview on the use of sprayed-on asbestos fireproofing. Data on Experimental measurements of emissions during application are presented.

58. I.J. Selikoff, W.J. Nicholson, A.M. Langer, "Asbestos Air Pollution," Arch Env. Health, 25:1 (July 1972).

Measurement of chrysotile content of ambient air in New York City showed levels of 1050 nanograms/m3, and lungs of New York residents examined at autopsy regularly showed chrysotile fibers. A determination of the health hazard has not been ascertained, however.

59. P.F. Holt and D.K. Young, "Asbestos Fibers in the Air of Towns," Atmospheric Environment, 7(5):481 (May 1973).

The air of several European cities (London, Reading, Rochdale, Dusseldorf, Prague, Johannesburg, and Reykjavik) has been sampled using millipore filters. Examination by electron microscope showed asbestos fibrils to be present in each city.

60. M.L. Newhouse, "Asbestos in the Workplace and the Community," Ann. Occ. Hygiene. 16(2):97 (August 1973).

Analysis of asbestos factory workers demonstrated that low or moderate exposure caused an excess cancer and mesothelioma rate. Conditions giving rise to neighborhood mesothelial tumors may not occur today, but the importance of adequate control is stressed.

61. J.S.P. Jones, "Pathological and Environmental Aspects of Asbestos-Related Associated Diseases," Medicine, Science and the Law, 14(3):152-8 (July 1974).

The percentage of autopsies in which asbestos was detected in post-mortem lung tissue is tabulated by geographical areas ranging from unpolluted rural districts (1-8%) to Finnish areas (39%) where the percentage of asbestos is known to be high.

62. P. Gross, "Is Short-Fibered Asbestos Dust a Biological Hazard?" Arch. Env. Health, 29:115 (August 1974).

It has been the finding of research laboratories in Germany, England, South Africa and the United States that short-fibered asbestos dust; i.e., less than five microns in length, is incapable of causing fibrosis or cancer. These studies involved injection of asbestos fibers into rats and guinea pigs.

63. M.L. Warnock and A. Churg, "Association of Asbestos and Bronchogenic Carcinoma in a Population with Low Asbestos Exposure," Cancer, 35:1236(1975).

Quantitative counts of ferruginous bodies were performed on digests of lungs from 100 control and 30 lung cancer patients. It was found that lung cancer had significantly higher levels, although only one patient was known to be occupationally exposed to asbestos. It is suggested that even extremely low levels of asbestos exposure may have a carcinogenic effect.

64. A.M. Rohl, A.M. Langer, I.J. Selikoff, W.J. Nicholson, "Exposure to Asbestos in the Use of Consumer Spackling, Patching, and Taping Compounds," Science, 189:551 (August 15, 1975).

An analysis of 15 representative samples of consumer spackling, patching and taping compounds has shown that five contained appreciable amounts of chrysotile or other asbestos minerals. Optical microscopic analysis of air samples obtained during the application of these materials showed concentrations in excess of 5f/cc.

65. W. Nicholson, A. Rohl, I. Weisman, Asbestos Contamination of the Air in Public Buildings, EPA Contract #68-02-1346, 1975. Summarized in Environmental Sensing and Assessment 1975, International Conference on Environmental Sensing and Assessment, Las Vegas, Vol. 2:29-6 (1976).

An investigation of asbestos use in high rise office buildings was conducted. Recommendations included development of a feasible and economical filtration system, procedures to minimize air contamination during building maintenance and procedures to remove asbestos where unacceptable contamination existed.

VIII. Measurement and Control of Asbestos

66. J.R. Lynch, H.E. Ayer, D.L. Johnson, "The Interrelationships of Selected Asbestos Exposure Indices," Amer. Ind. Hyg. Assoc. J. 31:598 (Sept.-Oct. 1970).

A discussion of the four major techniques of asbestos measurement leads to the conclusion that the phase contrast microscope is the preferred method.

67. Criteria for a Recommended Standard for Occupational Exposure to Asbestos, HEW Public Health Service, National Institute for Occupational Safety and Health, HSM 72-10267, Wash. D.C. 1972.

A review of previous medical literature relating to the occupational hazards of asbestos inhalation results in the recommendation of a 2 f/cc standard. This regulation became effective on July 1, 1976. (29 CFR 1910.1001).

68. Background Information on Development of National Emission Standards for Hazardous Air Pollutants, EPA Office of Air and Water Programs, APTD-1503, Research Triange Park, N.C. 1973.

This is the background paper for the initial EPA asbestos regulation of April 6, 1973 (38 FR 8820). Information on the environmental and economic impact of this standard is provided.

69. G.L. Lee and D.J. Smith, "Steelwork Insulated with Sprayed Crocidolite Asbestos: Controlling a Potential Hazard," Ann. Occ. Hygiene, 17:49 (August 1974).

Surveys of factory sites revealed the presence of crocidolite asbestos in a number of places. Dust sampling and visual evidence suggested a potential occupational health risk in one of these areas. A procedure for sealing the asbestos and insuring continued maintenance was adopted.

70. Background Information on National Emission Standards for Hazardous Air Pollutants - Proposed Amendments to Standards for Asbestos and Mercury, EPA Office of Air and Waste Management, EPA-450/2-74-005A, Research Triangle Park, N.C., October 1974.

Background information on the October 14, 1975 asbestos standard (40 FR 48292) is provided. Discussion of demolition and renovation of buildings, fabrication and disposal of asbestos wastes is included.

71. A.M. Langer, "Approaches and Constraints in Identification and Quantification of Asbestos Fibers," Env. Health Perspectives, 9:133 (December 1974).

Approaches and constraints to the identification and quantification of asbestos fibers are discussed. The major constraints is the signal to noise ratio; i.e., the amount of asbestos in a sample as compared to the amount of other particles

72. J.D. Gillian, P.A. Lemen, V.E. Archer, J.K. Wanner and J. Dement, "Morbidity and Mortality among Hard Rock Miners Exposed to an Asbestiform Mineral," Ann. N.Y. Acad. Sci. (in press) (reproduced in 40 FR 47656 (Oct. 9, 1975).

A review of the chest X-rays of 439 underground metal miners found three times the risk of malignant respiratory disease than expected. The fiber concentration averaged 6.24 f/cc.

73. Department of Labor, "Occupational Exposure to Asbestos - Notice of Proposed Rulemaking," 40 Federal Register 47652 (October 9, 1975).

A discussion of a proposed new standard (0.5 f/cc) is presented with an analysis of new evidence.

74. L. Bruckman, R.A. Rubino, "Asbestos-rationale Behind a Proposed Air Quality Standard," J. of the Air Pollution Control Association, 25:1027 (December 1975).

A standard based on the risk of mesothelioma is calculated by extrapolating data from the occupational environment. The level of 30 nanograms per cubic meter is suggested. It is projected to result in 150 nationwide fatalities.

75. R. Sawyer, "Yale Art and Architecture Building Asbestos Contamination," Environmental Sensing and Assessment - 1975, International Conference on Environmental Sensing and Assessment, Las Vegas, vol. 2:20-6 (1976).

A discussion of asbestos removal in the Yale Art and Architecture Building is presented. Quantitative measurement of asbestos levels showed some concentration exceeded the 2.0 f/cc limit. All building users were exposed to a level over that found in the ambient air of the community.